"PULSE OXIMETRY IN HYSTERECTOMY OPERATIONS"

A CLINICAL EVALUATION OF DIFFERENT ANAESTHETIC TECHNIQUES

THESIS

FOR

DOCTOR OF MEDICINE

(ANAESTHESIOLOGY)

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ANIL KUMAR VERMA

C E R T I F I C A T E

This is to certify that the work entitled "PULSE OXIMETRY IN HYSTERECTOMY OPERATIONS (A Clinical evaluation of different anaesthetic techniques)", which is being submitted as a thesis for M.D.(Anaesthesiology) by Dr. Anil Kumar Verma, has been carried out in the Department of Anaesthesiology, M.L.B. Medical College, Jhansi.

He has fulfilled the necessary stay in the department as required by the regulation of the Bundelkhand University, Jhansi.

Dated : 13.2.91

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C O N T E N T S

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INTRODUCTION

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PULSE OXIMETRY has been recommended as a standard of care for every general anaesthetic (Julien et al, 1989). This technique, virtually unknown in anaesthesia 5 years ago, has been so readily adopted for several reasons. The device provides valuable data regarding blood oxygenation and this information is obtained easily, continuously and non-invasively. Continuous assessment of arterial oxygenation is important in clinical management of critically ill or anaesthetised patients. Analysis of arterial blood gases is reliable but is invasive and only provides intermittent information likely to miss the transient but important episodes of hypoxaemia. Analysis of arterial blood gases and transcutaneous oxygen measurements both provide oxygen tension (PO_2) data from which the oxygen content and percentage of hemoglobin saturated with oxygen can be estimated. Arterial oxygen saturation of hemoglobin can be determined directly and continuously in vivo by using spectrophoto-electric oximetric techniques. The wavelength dependence of reduced versus oxyhemoglobin is evident from the prominent colour differences in spectral light absorbance of "red" oxyhemoglobin and "blue" reduced hemoglobin.

Pulse oximetry functions by positioning any pulsating arterial vascular bed between a two-wavelength light source and a detector. The pulsating vascular bed by expanding and relaxing, creates a change in the light path length that modifies the amount of light detected. The familiar plethysmograph wave form results. Because the detected pulsatile wave form is produced solely from arterial blood, using the amplitude at each wavelength and Beer's law allows exact beat-to-beat continuous calculation of arterial hemoglobin oxygen saturation with no interference from surrounding venous blood, skin, connective tissue or bone.

Hypoxemia has been a matter of concern not only in the operating room but also in the immediate post-operative period. However, clinical assessment of hypoxemia is rather difficult. The detection of cyanosis, the traditional sign of hypoxaemia, is very unreliable. The human eye is a poor judge of changes in skin colour, particularly in dark-skinned patients and under fluorescent lights. Cyanosis is only detectable when the arterial oxygen saturation (SaO_2) is below 80% (Nakatsuka et al, 1989). The recent introduction of "pulse oximetry" has provided a continuous, non-invasive, real time method to detect SaO_2 intra-operatively and post-operatively. Indian women frequently suffer from anaemia. The risk factors of intra-operative and post-operative hypoxemia in "Hysterectomy" operations with pre-existing anaemia under different

techniques of anaesthesia may be injurious to the patients.

A paper published as early as 1951 in "ANAESTHESIOLOGY" concluded prophetically that "On many occasions this instrument has detected anoxemia when observations of pulse, blood pressure and colour of the patient and peripheral vascular tone have shown no abnormalities".

The clinical utility of the non-invasive oximeter in the operating room was discovered in 1980s by William New, an anaesthesiologist at Standford University, realising that a continuous, non-invasive monitor of oxygenation would be useful to anaesthesiologists.

Severe arterial hypoxaemia may occur even during the most meticulously administered anaesthetic. Prolonged moderately severe hypoxia may be associated with pre-existing anaemia and respiratory disease. Anaemia is very frequently associated with Indian women and anaesthetic practice more so when major surgery like hysterectomy operations.

It was therefore, thought worthwhile to evaluate changes in oxygen saturation by Pulse Oximetery intra-operatively and in immediate post-operative period in hysterectomy operations planned under different anaesthetic techniques.

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

History :

As with any new techniques, many people contributed to the development of usable in vivo oximeters. Two early researchers who stand out are Carl Matthes and Glen Millikan. Matthes is often considered the faterh of oximetry. Between 1934 and 1944 he published a series of articles investigating oxygen transport to tissue by light transmission techniques.

In 1935 Kramer also demonstrated that the transmission of red light through tissue was dependent on oxygen saturation but since he employed only one wave-length of light, this method only measured trends in saturation.

In 1935 Matthes built the first device that continuously measured human blood oxygen saturation in vovo by transilluminating tissue. He used two wavelengths of light, one that was sensitive to changes in oxygenation and another that was not. The second wavelength, in the impaired range, was used to compensate for changes in tissue thickness, hemoglobin content and light intensity. This device could follow trends in saturation but was difficult to calibrate.

J.R. Squire, in Great Britain, developed a similar device that was calibrated by compressing the tissue to eliminate the blood. This same calibration technique was later adopted in the first oximeters used in the operating room.

In the early 1940s, Glen Millikan coined the term "oximeter" to describe a light-weight device he developed for aviation research.

Later in the 1940s, oximeters similar to Millikan's were used by Earl Wood and others in the operating room, where they were noted to detect significant desaturations even during routine anaesthetics.

In the United States in 1942, Glen Millikan and colleagues developed a small ear oximeter for use in aviation research.

A paper published as early as 1951 in ANAESTHESIOLOGY concluded prophetically that "on many occasions this instrument has detected anoxemia when observations of pulse, blood pressure and color of the patient, and peripheral vascular tone have shown no abnormalities". These findings were consistent with the classic work of Comore et al (1947) documenting the unreliability of cyanosis for the detection of anoxemia.

In its initial clinical development, the ear oximeter had several limitations. It was a delicate

instrument that required a technician to operate and maintain. The ear-piece was large, difficult to position and produced enough heat to cause second degree burns on the pinna. Furthermore, it required calibration on each patient prior to use.

During the 1950s, Earl Wood devised a modification of the Millikan ear piece that was used in many clinical and laboratory investigations. Although the ear oximeter showed promise in some settings, it was still considered a research tool.

In the 1960s, Robert Shaw developed a self-calibrating eight-wavelength ear oximeter that was produced by Hewlett-Packard. Although cumbersome and expensive, this device became the standard for oximetry because of its accuracy.

In the 1970s, Hewlett-Packard marketed the first self calibrating ear oximeter. This device used eight wavelengths of light to determine hemoglobin saturation. Hewlett-Packard's oximeter also used the method of heating the ear to "arterialize" the capillary blood. This oximeter quickly became a standard clinical and laboratory tool in pulmonary medicine.

In the mid 1970s, Takuo Aoyagi, an engineer working for Nihon Kohden Corporation, made an ingenious discovery regarding oximetry. He was developing a method

to estimate cardiac output semi non-invasively by detecting the wash-out curve of dye injected into a peripheral vein as it perfused the ear. This washout curve was measured in the ear with a red and infra-red light densitometer similar to the Milliken ear oximeter. He noticed that his washout curves contained pulsations due to the arterial pulse in the ear. To more easily analyze the dye washout curve, he subtracted these pulsations from the curve, and in doing so he discovered that the absorbance ratio of the pulsations at the two wavelengths changed with arterial hemoglobin saturation. He soon realised that he could build an ear oximeter that measured arterial hemoglobin saturation without heating the ear by analysing pulsatible light absorbances. This first pulse oximeter, developed by Nihon Kohden, used filtered light sources similar to Milliken's ear oximeter. The device was evaluated clinically in the mid 1970s, and marketed with little success.

In the late 1970s, Scott Wilber in Boulder, Colorado, developed the first clinically accepted pulse oximeter by making two modifications of the Nihan Koholen method. First, he produced a light-weight sensor by using light emitting diodes (LEDs) as light sources and photo diodes as detectors.

In the year 1975 Nakajima and colleagues introduced the pulse oximeter. By analysing the ratio of the pulse-added absorbances of the red and infra-red light,

this method allowed accurate determination of hemoglobin saturation with only two wave-lengths of light on various tissue thicknesses and skin colors. This device, developed by Minolta, used fiberoptics to transmit the light signals to and from a finger sensor.

In the early 1980s, these fiberoptics were cumbersome, and Minolta's monitor was quickly replaced by pulse oximeters developed by BTI, Biox Corporation of Boulder, Colorado, and was successfully marketed to pulmonary function laboratories.

The clinical utility of the non-invasive oximeter in the operating room was re-discovered in the 1980s by William New, an anaesthesiologist at Stanford University. Realising that a continuous, non-invasive monitor of oxygenation would be useful to anaesthesiologists, New developed and marketed a pulse oximeter to this group. The Nellcor model N 100 had by 1985 become almost synonymous with the term "pulse oximeter".

THE PHYSICS AND PHYSIOLOGY OF PULSE OXIMETRY

BEER'S LAW :

In the 1930s, Matthes used spectrophotometry to determine hemoglobin oxygen saturation. This method is based on the Beer-Lambert Law, which relates the concentration of a solute to the intensity of light transmitted through a solution.

$$I_{trans} = I_{in}e^{-A} \quad (1)$$

$$A = DC_E \quad (1a)$$

where I_{trans} = intensity of transmitted light

I_{in} = intensity of incident light

A = absorption

D = distance light is transmitted through the liquid (path length)

C = concentration of solute (hemoglobin)

E = extinction coefficient of the solute (a constant for a given solute at a specified wavelength).

Thus, if a known solute is in a clear solution in a cuvette of known dimensions, the solute concentration can be calculated from measurements of the incident and transmitted light intensity at a known wave length. The extinction coefficient E is a property of light absorption for a specific substance at a specified wave-length. In a one-component system, the absorption A is the product of the path-length, the concentration and the extinction coefficient, equation 1a. If multiple solutes are present, A is the sum of similar expressions for each solute. The extinction coefficient can vary dramatically with the wave-length of the light (Fig. 1).

HEMOGLOBIN EXTINCTION CURVES

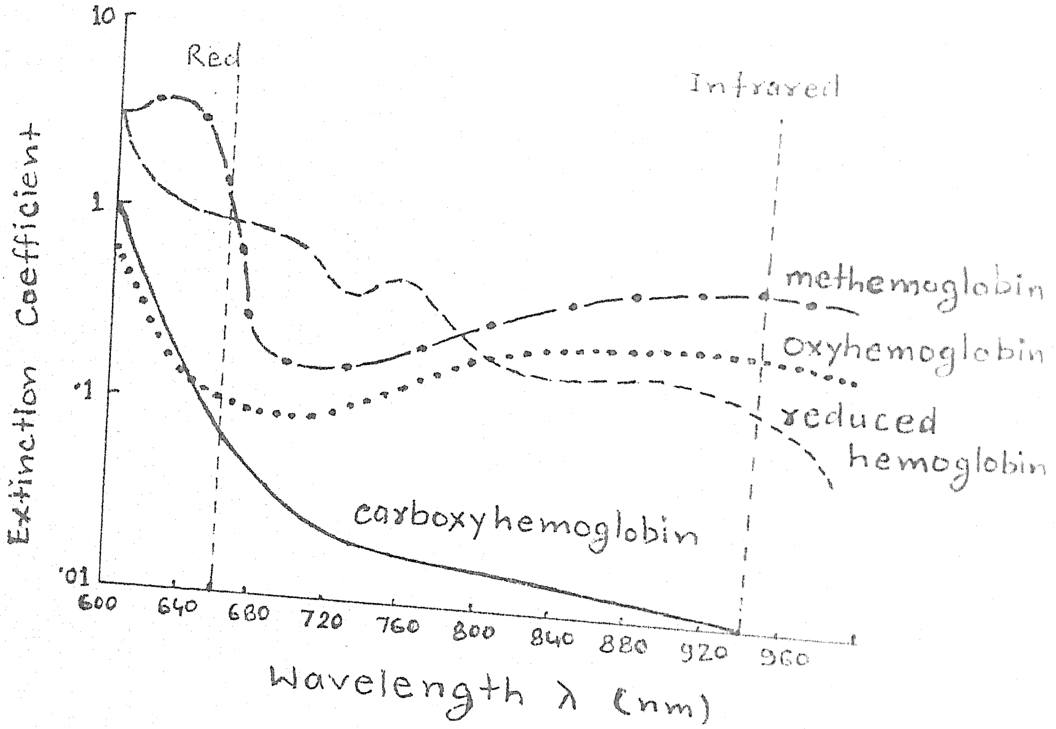


Fig: 1 : Transmitted light absorbance spectra of four hemoglobin species: oxyhemoglobin, reduced hemoglobin, carboxyhemoglobin, and methemoglobin. Adapted from Barker SJ and Tremper KK: Pulse Oximetry: Applications and limitations, Advances in Oxygen Monitoring, International Anaestesiology Clinics Boston, Little, Brown and Company , 1987, pp. 155-175.

Laboratory oximeters use this principle to determine hemoglobin concentration by measuring the intensity of light transmitted through a curvette filled with a hemoglobin solution produced from lysed red blood cells. For Beer's law to be valid, both the solvent and the curvette must be transparent at the wavelength used, the light path length must be known exactly, and no absorbing species can be present in the solution other than the known solute. It is difficult to fulfil these requirements in clinical devices, therefore, each instrument theoretically based on Beer's law also requires emperical corrections to improve accuracy.

HEMOGLOBIN SATURATION DEFINITIONS

Adult blood usually contains four species of hemoglobin : oxyhemoglobin (O_2Hb), reduced hemoglobin (Hb), methemoglobin (Met Hb) and carboxyhemoglobin (CO Hb) (Fig. 1). The last two species are in small concentrations, except in pathologic condition. There are several definitions of hemoglobin saturation. Historically, "Oxygen saturation" was first defined as the oxygen content expressed as a percentage of the oxygen capacity. The oxygen content (cc of oxygen per 100 cc of blood) was measured volumetrically by the method of Van Slyke and Neill (1924). The oxygen capacity was defined as the oxygen content after the blood sample had been equilibrated

with room air (158 mm Hg oxygen at sea level). By the above definition of oxygen saturation, the two form of hemoglobin that do not bind oxygen (CO Hb and Met Hb) are not included. This is the origin of what is now referred to as "functional hemoglobin saturation", defined as (Severinghaus, J.W., personal communication) :

$$\text{Functional } \%\text{a } O_2 = \frac{O_2 \text{ Hb}}{O_2 \text{ Hb} + \text{Hb}} \times 100\% \qquad (2)$$

with the advent of multi-wavelength oximeters that can measure all four species of hemoglobin, "fractional saturation" has been defined as the ratio of oxyhemoglobin to total hemoglobin :

$$\text{Fractional } \%\text{a}O_2 = \frac{O_2 \text{ Hb}}{O_2\text{Hb} + \text{Hb} + \text{CO Hb} + \text{Met Hb}} \times 100\% \qquad (3)$$

The fractional hemoglobin saturation is also called the "oxyhemoglobin fraction" or "oxyhemoglobin %".

When oximetry is used to measure hemoglobin saturation, Beer's Law must be applied to a solution containing four unknown species : O_2Hb, Hb, CO Hb, and Met Hb. Expanding equation 1a to a four-component system results in an absorption given by :

$$A = D_1C_1E_1 + D_2C_2E_2 + D_3C_3E_3 + D_4C_4E_4 \qquad 1(b)$$

The subscripts 1 through 4 correspond to the four hemoglobin species. If the path lengths are the same, then D can be factored out :

$$A = D\,(C_1E_1 + C_2E_2 + C_3E_3 + D_4E_4) \qquad 1(c)$$

The extinction coefficients E_1 through E_4 are constants at a given wavelength (Fig. 1). The absorption defined in equation 1c is determined from equation 1 by measuring the incident and transmitted light intensities. From equation 1c, we see that four wavelengths of light are needed to produce four equations to solve for the unknown concentrations C_1 through C_4. If CO Hb and Met Hb were not present their contributions to the absorption would be zero and functional hemoglobin saturation could be determined by a two-wavelength oximeter (two equations and two unknowns). If two wavelengths existed for which the extinction coefficients for CO Hb and Met Hb were zero, then these absorption terms would again be zero and a two wavelength oximeter could measure functional saturation. Unfortunately, as illustrated in Fig. 1, the extinction coefficients for CO Hb and Met Hb are not zero in the red and infra-red range, and their presence will, therefore, contribute to the absorption. Even though the definition of functional hemoglobin saturation involves only two hemoglobin species (O_2 Hb and Hb), when Met Hb and CO Hb are present, four wavelengths are required to determine either functional or fractional hemoglobin saturation.

<u>PULSE OXIMETRY</u>

Non-invasive oximeters measure red and infra-red light transmitted through a tissue bed, effectively using

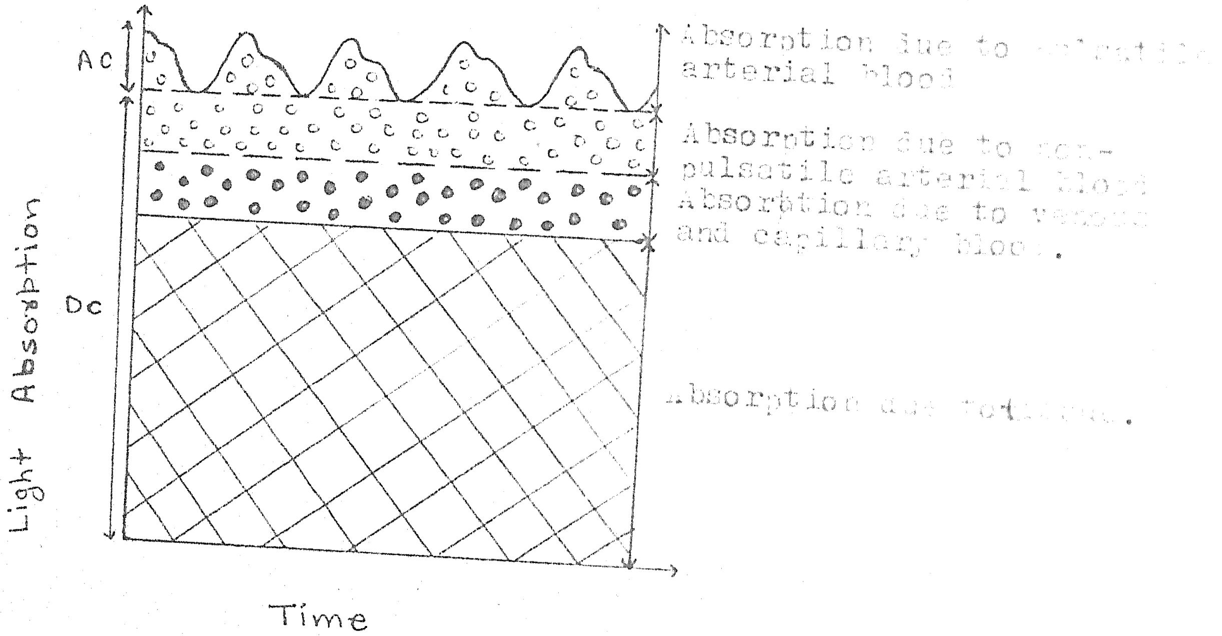
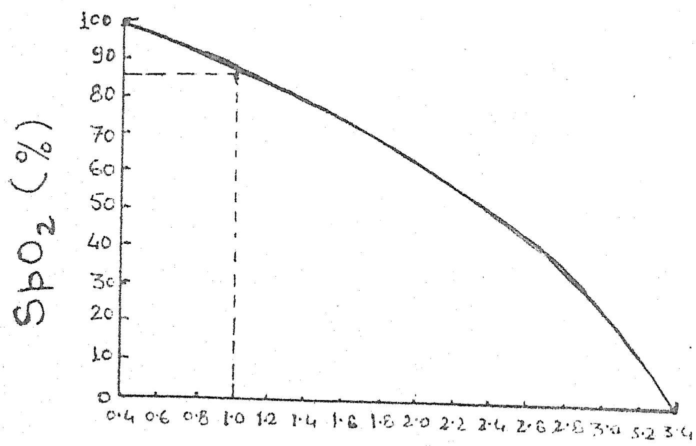


Fig.2: This figure schematically illustrates the light
absorption through living tissue. Note that the AC
signal is due to the pulsatile component of the arterial
blood while the DC signal is comprised of all the
nonpulsatile absorbers in the tissue;nonpulsatile
arterial blood, venous and capillary blood, and all other
tissues. Adapted from Ohmeda Pulse Oximeter Model
3700 Service Manual, 1986, P.22.

the finger or ear as a curvette containing hemoglobin. There are several technical problems in accurately estimating SaO_2 by this method. First, there are many absorbers in the light path other than arterial hemoglobin, including skin, soft tissue, and venous and capillary blood. The early oximeters subtracted the tissue absorbance by compressing the tissue during calibration to eliminate all the blood, and using the absorbance of bloodless tissue as the base-line. These oximeters also heated the tissue to obtain a signal related to arterial blood with minimum influence of venous and capillary blood.

Pulse oximeters deal with the effects of tissue and venous blood absorbances in a completely different way. (Fig. 2). Schematically illustrates the series of absorbers in a living tissue sample. At the top of the figure is the pulsatile or AC component, which is attributed to the pulsating arterial blood. The baseline or DC component represents the absorbances of the tissue bed, including venous blood, capillary blood, and non-pulsatile arterial blood. The pulsatile expansion of the arteriolar bed produces an increase in path length (equation 1b), thereby increasing the absorbance. All pulse oximeters assume that the only pulsatile absorbance between the light source and the photodetector is that of arterial blood. They use two wavelengths of light : 660 nanometers (red) and 940 nanometers (near infra-red). The pulse oximeter first determines the AC component of absorbance at each wavelength



$$R = \frac{AC_{660}/DC_{660}}{AC_{940}/DC_{940}}$$

Fig.3: This is a typical pulse oximeter calibration curve . Note that the SaO_2 estimate is determined from the ratio(R) of the pulse-added red absorbance at 660 namometres to pulse-added infrared absorbance at 940 nanometers. The ratios of red to infrared absorbances varyfrom approximately 0.4 at 100% saturation to 3.4 at 0% saturation. Note that the ratio of red to infrared absorbance is one at a saturation of approximately 85% . This curve can be approximately determined on a theoretical basis but for accurate predictions of SpO_2 , experimental data are required. Adapted from JA Pologe;Pulse oximetry:Technical aspects of machine design, International Anaesthesiologyclinics. Advances in Oxygen Monitoring. Edited by Tremper KK, Barker SJ. Boston, Little, Brown and Company , 1987 , P.142.

and divides this by the corresponding DC component to obtain a "pulse-added" absorbance that is independent of the incident light intensity. It then calculates th ratio (R) of these pulse-added absorbances, which is empirically related to SaO_2 :

$$R = \frac{AC_{660} / DC_{660}}{AC_{940} / DC_{940}} \qquad (4)$$

Fig. 3 is an example of a pulse oximeter calibration cur The actual curves used in commercial devices are based o experimental studies in human volunteers. Note that whe the ratio of red to infra-red absorbance is one, the saturation in approximately 85%. This fact has clinical implications to be discussed later.

It is a fortuitous coincidence of technology and physiology that allowed the development of solid-state pulse oximeter sensors. Light emitting diodes (LEDs) are available over a relatively narrow range of the electro-magnetic spectrum. Among the available wavelengths are some that not only pass through skin but also are absorbed by both oxyhemoglobin and reduced hemoglobin. For best sensitivity, the difference between the ratios of the absorbances of O_2 Hb and Hb at the two wavelengths should be maximized. As we see in Fig. 1, at 660 nm, reduced hemoglobin absorbs about ten times as much light as oxyhemoglobin (Note that the extinction coefficients ar

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plotted on a logarithmic axis). At the infra-red wavelength of 940 nm, the absorption coefficient of O_2Hb is greater than that of Hb.

Engineering Design and Physiologic Limitations :

Although the theory on which pulse oximetry is based is relatively straight-forward, the application of this theory to the production of a clinically useful device involves a significant engineering effort. This section will present in general terms the clinical and physiologic problems of oximeter design and their engineering solutions. This discussion is divided into four areas :

Dyshemoglobins and dyes,

LED center wavelength variability,

Signal artifact management, and

Accuracy and response.

Dyshemoglobins and Dyes :

Being two-wavelength devices, pulse oximeters can deal with only two hemoglobin species. As noted above, this would be adequate to measure functional SaO_2 if Met Hb and CO Hb did not absorb red or infra-red light at the wavelengths used. Unfortunately, this is not the case, and therefore both Met Hb and CO Hb will cause errors in the pulse oximeter reading. It is not intuitively obvious how a pulse oximeter will behave in the presence of

dyshemoglobins. With respect to carboxyhemoglobin, we can gain some insight from the extinction curves of Fig. 1. In the infra-red range (940 nm), CO Hb absorbs very little light; whereas, in the red range (660 nm), it absorbs as much light as does O_2 Hb. This is clinically illustrated by the fact that patients with carboxyhemoglobinemia appear red. Therefore, to the pulse oximeter, CO Hb looks like O_2Hb at 660 nm; while, at 940 nm CO Hb is relatively transparent. The effect of CO Hb on pulse oximeter values has been evaluated experimentally in dogs. In this study, the pulse oximeter saturation (Sp O_2) was found to be given approximately by :

$$SpO_2 = \frac{O_2Hb + 0.9 \times CO\ Hb}{Total\ Hb} \times 100\% \qquad (5)$$

The effects of methemoglobinemia on pulse oximetry are also partially predictable from the extinction curves (Fig. 1). Met Hb has nearly the same absorbance as reduced hemoglobin at 660 nm, while it has a greater absorbance than the other hemoglobins at 940 nm. This is consistent with the clinical observation that methemoglobinemia produces very dark, brownish blood. Thus, it would be expected to produce a large pulsatile absorbance signal at both wavelengths. The effect of Met Hb on pulse oximeter readings has also been measured in dogs. As methemoglobin levels increased, the pulse oximeter saturation (Sp O_2) tended towards 85% and eventually became almost independent

of the actual SaO_2. In other words, in the presence of high levels of Met Hb, SpO_2 is erroneously low when SaO_2 is above 85% and erroneously high when SaO_2 is below 85%. This may be explained by the fact that Met Hb causes a large pulsatile absorbance at both wavelengths, thereby adding to both the numerator and denominator of the absorbance ratio R (equation 4) and forcing this ratio towards unity. As shown in Fig. 3, an absorbance ratio of one corresponds to a saturation of 85% on the calibration curve, pulse oximeter error during methemoglobinemia has also been confirmed in a clinical report.

In neonatal blood, a fifth type of hemoglobin is present, fetal hemoglobin (HbF). HbF differs from adult Hb in the amino acids sequences of two of the four globin sub-units. Adult Hb has two alpha and two Beta-globin chains, while HbF has two alpha and two f chains. This difference in globin chains has little effect on the extinction curves and therefore should not affect pulse oximeter readings. This is indeed fortunate because the fraction of HbF present in neonatal blood is a function of gestational age and can not be accurately predicted. HbF does produce a small error in (in vitro) laboratory oximeters; O_2 HbF may be interpreted as consisting partially of COHb.

The absorbance ratio R (equation 4) may be affected by any substance present in the pulsatile blood that absorbs light at 660 or 940 nm and was not present

in the same concentration in the volunteers used to generate the calibration curve (Fig. 3). Intravenous dyes provide a good example of this principle. Scheller et al (1986) evaluated the effects of bolus doses of methylene blue, indigo carmine, and indocyanine green on pulse oximeters in human volunteers. They found that methylene blue caused a fall in SpO_2 to approximately 65% for 1-2 min. Indigo carmine produced a very small drop in saturation, while indocyanine green had an intermediate effect. The detection of intravenous dyes by pulse oximeters should not be surprising, because it was this effect that led Aoyagi to the invention of pulse oximetry.

<u>LED Center Wavelength Variability</u> :

The LEDs used in pulse oximeter sensors are not ideal monochromatic light sources; there is a narrow spectral range over which they emit light. The center wavelength of the emission spectrum varies even among diodes of the same type from the same manufacturer. This variation can be ± 15 nanometers. As seen in Fig. 1, a shift in LED center wavelength will change the measured extinction coefficient and thus produce an error in the saturation estimate. This source wavelength effect will be greatest for the red (660 nm) wavelength, because the extinction curves have a steeper slope at this wavelength. Manufacturers have found two approaches to this problem. Some test all the LEDs and reject those that are out of

their specified wavelength range, e.g. 660 ± 5 nanometers. This is expensive due to the number of LEDs rejected; i.e. narrower acceptable range yields improved accuracy but also more rejected LEDs. Alternatively, other manufacturers program the pulse oximeter to accept several ranges of LED center wavelengths for both the red and infra-red, allowing the device to correct internally for different wavelengths. This permits the manufacturer to use more of the available LEDs, but also requires a more sophisticated device with a mechanism for identifying the sensor LED wavelengths to the pulse oximeter. Incompletely compensated LED frequency variation will not change the pulse oximeter's ability to trend saturation changes, but will produce probe to probe variability in the absolute measurement of $Sa\ O_2$.

Signal Artifact Management :

Probably the most difficult engineering problem in pulse oximeter design is the identification of the "ripple" absorbance pattern of the arterial blood in a "sea" of electromagnetic artifact. Artifact has three major sources : ambient, light, low perfusion (low AC/DC signal), and motion (large AC/DC signal). All of these result in poor signal-to-noise ratio.

The photodiodes used in the sensor as light defectors cannot discriminate one wavelength of light from another. Therefore, the detector does not know whether received light originates from the red LED, the infra-red

LED, or the room lights. This problem is solved by alternating the red and infra-red LED. The red LED is turned on first and the photodiode detector produces a current resulting from the red LED plus the room lights. Next, the red LED is turned off and the infra-red LED is turned on, and the photodiode signal represents the infra-red LED plus the room lights. Finally, both LEDs are turned off and the photodiode generates a signal from the room lights alone. This sequence is repeated hundreds of times per second. In this way, the oximeter attempts to eliminate light interference even in a quickly changing background of room light. Some fluctuating light sources can cause problems in spite of this clever design. Clinically, ambient light artifact can be minimized by covering the sensor with an opaque shield.

Another engineering problem is that of low AC-to-DC signal ratio. When a small pulsatile absorbance signal is detected the pulse oximeter will amplify that signal and estimate the saturation from the ratio of the amplified absorbances. The pulse oximeter can thereby estimate saturation values for a wide range of patients with differing pulsatile absorbance amplitudes. Unfortunately, as with a radio receiver, when a weak signal in amplified, the background noise (static) is also amplified. At the highest amplifications (which can be as much as a billion times), the pulse oximeter may analyze this noise signal and generate an Sp O_2 value from it. This problem could

be demonstrated in early pulse oximeters by placing a piece of paper in the sensor between the photodiode and the LED. Most early models would amplify the background noise in searching for a pulse until they eventually displayed a pulse and saturation value. To prevent this type of artifact, manufacturers have now incorporated minimum values for signal-to-noise ratio, below which the device will display no Sp O_2 value. Some oximeters also display a low signal strength error message, and some display a plethysmographic wave for visual identification of noise.

Several studies have examined the effect of low perfusion on pulse oximeter estimates. Animal experiments have demonstrated that, during hemorrhagic shock, pulse oximeters may under-estimate saturation or lose signal altogether. In one clinical study of pulse oximeter, accuracy in the critically ill under a wide range of hemodynamic conditions, extremes in systemic vascular resistance were associated with loss of signal or decreased accuracy. In these and most other studies of pulse oximeter accuracy, data were collected only when the pulse oximeter heart rate equalled the EKG heart rate. It has been assumed that this is a necessary condition for accuracy because it implies that the pulse oximeter is detecting pulses produced by heart-beats.

Since the device automatically increases its amplification as the pulse signal decreases, the pulse oximeter display should be relatively insensitive to changes in perfusion. Nevertheless, several clinical studies have used the pulse oximeter to assess the adequacy of peripheral perfusion. One study even employed this device to evaluate perfusion in reimplated extremities. As with any plethysmograph, the pulse oximeter will detect a complete loss of peripheral blood flow, as has been illustrated by Lawson et al (1987). They determined the peripheral blood flow lower limit at which a pulse oximeter ceased detecting pulses. The blood flow was assessed at the finger by a laser-doppler flow probe as a blood pressure cuff was inflated. The pulse oximeter stopped detecting pulses when blood flow had decreased to 8.6% of its control value, which occurred when the pressure cuff was inflated to 96% of the control systolic pressure. When the tourniquet was slowly released from full occlusion, the pulse oximeter regained a pulse and saturation value when blood flow was only 4% of the baseline. This experiment demonstrates the effectiveness of the pulse oximeter in detecting and amplifying small pulse signals to estimate arterial hemoglobin saturation. This experimental model is not analogous to clinical shock, for as the blood pressure cuff is progressively inflated, there is a progressive increase in the venous blood volume. Theoretically, this increase in venous blood should not influence the pulse oximeter because it is non-pulsatile.

Patient motion (large AC/DC signal) may be the most difficult artifact to eliminate. Motion artifact rarely causes difficulties in the operating room, but in the recovery room and intensive care unit, it can make the pulse oximeter nearly useless. Engineers have tried several approaches to this problem, beginning with the signal averaging time. If the device averages its measurements over a longer time period, the effect of an intermittent artifact will be lessened. This also slows the response time to an acute change in Sa O_2. Most pulse oximeters allow the user to select one of several time averaging modes. In addition, the designer can use sophisticated algorithms to identify and reject spurious signals. These algorithms may assess the AC-to-DC signal ratio, or they may check the validity of the saturation estimate by calculating its rate of change. For example, if the saturation estimate changes from 95% to 50% in one-tenth of a second, this sudden change may not be averaged into the displayed Sp O_2, or it may be given a lower weighting factor. As stated earlier, these artifact rejection schemes may also affect the accuracy and response time of the pulse oximeter.

Accuracy and Response :

There are both technologic and physiologic limitation to the accuracy of a pulse oximeter. The SpO_2

value is only as accurate as the empirical calibration curve programmed into the device, which, in turn is only as accurate as the in vitro laboratory oximeter used to generate it. The instrumentation laboratories model 282 Co-oximeter claims an accuracy of ± 1% for fractional saturation (± 2 standard deviations) when the pH is 7.0 - 7.4, Met Hb is 0 - 10% and the total hemoglobin is 12 - 16 gms/dl.

Before reviewing studies that are intended to determine pulse oximeter accuracy, we should discuss some problems in the statistical interpretation of accuracy data. These studies are referred to by statisticians as "methods comparison studies". A methods-comparison study was two methods to measure the same variable. One method is usually a new technique (in this case, pulse oximetry), and the other is a "gold standard" (in this case, in vitro saturation measurements from arterial blood samples). Bearing in mind that both methods have uncertainty, we wish to know what error to expect if the new method is compared to the standard. In the medical literature, the data analysis usually includes a correlation coefficient (r) with a P value, and a linear regression slope and intercept. Unfortunately, this is not the most informative statistical analysis for methods-comparison studies. The correlation coefficient is not a measure of agreement : it is a measure of association. We know that

pulse oximeter SpO_2 values and SaO_2 values are highly associated and we therefore expect a correlation coefficient that is significant. This does not tell us whether one measure of saturation can be used in place of the other, or what degree of confidence we should have in the new measure.

As an alternative, Altman recommends calculating the mean and standard deviation of the difference between the two methods of measurement. The mean of the difference is called the 'bias' and the standard deviation is often referred to as the "precision". The bias will show a systematic over-estimate or under-estimate of one method relative to the other, while the precision will represent the variability of "random error". If these systematic and random errors are clinically accepted table, then one method can be replaced by the other.

Unfortunately, in the pulse oximetry literature, many authors provide only correlation coefficient and linear regression analysis. It is difficult to compare their results in terms of measurement accuracy without bias and precision values. Most manufacturers claim that their pulse oximeters are accurate to within ± 2% (SD) from 70% to 100% saturation and ± 3% (SD) from 50% to 70% saturation, with no specified accuracy below 50% saturation. This implies that, for SaO_2 above 70%, approximately 68%

of the data will fall within $\pm$ 2% of a line of identity, and 95% of data will fall within $\pm$ 4% ($\pm$ 2 S.D.).

In reviewing the pulse oximetry literature, two additional points should be kept in mind. First, some of these studies were carried out in the healthy adult volunteer subjects, while others were conducted on patients in a variety of clinical settings. The studies using healthy volunteers were performed under optimal conditions, while the clinical studies were done in a variety of less than optimal conditions. Second, since these devices are empirically calibrated, the algorithm programmed into each oximeter undergoes a series of revisions that affect the accuracy and response characteristics. Table 1 summarizes the results from twelve studies : five in adult volunteers, three in adult patients, and two each in pediatric and neonatal patients. The data from each of these studies were analysed differently by the authors. Most consistently presented are correlation coefficients and regression slopes and intercepts. This is sometimes accompanied by a standard error of the estimate (SEE or Syx), which is the standard deviation of Y values about the regression line.

Experimental studies on early models of the Nellcor N 100 and the Ohmeda Biox II showed good agreement under steady state conditions when the saturation was 75% or greater (Table 1). Chapman et al noted that in this SaO_2 range the bias was only 0.09%. For SaO_2 less than 75%,

they found increasing over-estimation by the pulse oximeter. Between 50% and 60% SaO_2, there was a positive bias of 11.2% whereas between 70% and 75% the bias was 3.88%.

Two recent studies are of particular interest because they evaluated pulse oximeter accuracy during deep desaturation and also measured response times to rapid desaturation and resaturation. Both studies revealed errors in some manufacturers' calibration algorithms. This prompted these manufacturers to revise their algorithms and their devices were subsequently revaluated. This emphasize again the importance of specifying the software revision employed in any pulse oximeter study. Unfortunately, most reports do not specify the software revision (Table 1). Kagle et al (1987) evaluated the Ohmeda 3700 (KJ1 software) and the Nellcor N 100 in a volunteer study and found 99% prediction limits of ± 8% over a saturation range of 60 - 100%. Since 99% prediction limits are ± 3 SD, this implies a standard deviation of ± 2.7%, not far from manufacturers specifications. These authors also measured the time for 50% recovery of resaturation from a hypoxic state. With the pulse oximeter set on the "fast" (3_s) averaging mode, the ear probe showed resaturation more quickly than the finger probe (6_s versus 24_s).

Severinghaus and Naifeh (1987) published an interesting volunteer study comparing seven different pulse oximeters during severe desaturation. They also measured response times for both ear and finger probe desaturation and resaturation. This study did not determine accuracy over a range of steady-state saturation, but rather during a sudden, brief desaturation to an SaO_2 of 40 - 70% (Table 1). The authors noted significant variations in bias and precision among manufacturers as well as among subjects. The bias varied from $\pm$ 13% to 9%, with a precision as high as 16%. They also found that ear sensors were usually more accurate than finger sensors. This difference in accuracy could be a result of the unsteady nature of this experiment. The SpO_2 response times were again much faster for ear probes than for finger probes. The T ½ for the ear probe during desaturation ranged from 24 to 35.1_s. This differing response time is presumably due to different perfusion time constants for the ear and finger circulation. The response to resaturation was faster than to desaturation. One problem with this study that may limit the comparison of the devices is that the signal averaging times of the monitors were not the same. This would affect their response time to transients, and may also affect their accuracy during brief, deep desaturation. The SpO_2 values from the finger probes were still falling when the expired oxygen level and ear sensor SpO_2 had already shown resaturation. Therefore, some oximeters may

TABLE 1 : Pulse Oximeter Experimental and Clinical Accuracy Data.

Reference	Manufacturer	r	s	i	N	SEE% (Syx)	Range % high-low	Bias ± Prec.
Experimental Studies in Adult Volunteers :								
Yelderman	N-100	.98	1.03	-2.33	79	1.83	98-65	
Chapman	Biox II	.96	.79	17.9	117	2.72	100-54	
Kagle	Ohmeda 3700 (XJ1)	.99	.96	4.59	48	2.7*	99-60	
	N-100	.99	.96	5.36	48	2.7*	99-60	
Severinghaus	N-100				60		70-40	6.6 ± 10.8
	N-200				60		70-40	-4.5 ± 8.2
	Ohmeda 3700				60		70-40	2.7 ± 5.8
	CR (.28)				60		70-40	1.4 ± 5.9
	PC (1600)				60		70-40	0.0 ± 3.5
	NO (3.3)				120		70-40	1.1 ± 5.4
	NO (7)				36		70-40	-2.9 ± 5.2
	Datex				59		70-40	-1.6 ± 5.4
Nickerson	Ohmeda 3700				165		100-65	-2.6 ± 2.1
	CR				165		100-65	-1.0 ± 2.8
	N-100				165		100-65	-0.4 ± 1.7
	NO				165		100-65	-1.0 ± 1.6
Clinical Studies in Adult Patients :								
Tremper ǂ	Biox III	.57	.93	5.22	383	3.09	100-81	1.4 ± 3.1
Mihm	N-100	.96	.97	1.51	131		100-56	
Cecil	Ohmeda 3700	.83	.95	.42	333		100-62	-0.31± 2.46
	N-100	.80	.78	21.2	330		100-62	0.59± 3.02
Clinical Studies in Pediatric Patients :								
Fait	N-100	.89	1.05	-6.56	192		100-70	
Boxer	N-100	.95	1.01	0.15	108		95-35	- .87± 3.7
Clinical Studies in Neonatal Patients :								
Mok	N-100	.84	.65	27.8	27		100-43	1.4
Durand		.86	.68	29.6	108	2	100-78	- .2 ± 2.5

r = Correlation coefficients, s = Linear regression slopes. i = intercepts. N = No.of data pairs.
SEE% (Syx) = Standard error. Bias = mean difference between SpO_2 and SaO_2.
Prec. = standard deviation of the differences.

Contd.....

All manufacturers specified accuracy are similar, 1 SD = ± 2%, 100% to 70-80%, 1 SD = ± 3%, 70-80% to 50%, and unspecified $\angle$ 50%. Manufactures: N-100 and N-200 (Nellcor); Biox II, Biox III, and Ohmeda 3700 (Ohmeda); CR (Criticon); PC 1600 (Physio Control); NO (Novametrix); MQ (Marquest); and Datex. The software revision is in parenthesis following the manufacturer abbreviation when this information was provided in the referenced study. (Nellcor N-100 Technical Manual. Nellcor Corporation, Hayward, CA; Ohmeda 3700 Pulse Oximeter Technical Manual. Ohmeda Division of BOC, Boulder, CO ; Novametrix 500 Pulse oximeter Technical Manual. Novametrix Medical Equipment, Wallingford, CT).

* These values of Syx are determined from the authors 99% confidence intervals.

† The SpO_2 data were collected in patients with pulmonary artery catheters for simultaneous cardiac output determinations. Therefore, these patients were probably more critically ill than those in the other studies.

have not reached equilibrium at steady-state desaturated SpO_2 values due to their longer averaging time.

The most recent volunteer study examined the accuracy of four pulse oximeters over a range of saturation from 65 to 100% (Table 1). This study also compared the results of three bench oximeters and SaO_2 calculated from a blood gas analyzer using the multi-wavelength IL 282 co-oximeter as the reference. Two of the bench oximeters (which employed two wavelengths) yielded bias and precision values of -4.2 ± 2.3% and +1.8 ± 0.7%. The calculated SaO_2 values from the blood gas machine were of similar accuracy -1.8 ± 2.4%. The accuracies of the pulse oximeters compared favourably to those values (Table 1).

Among the clinical studies on adult patients, Tremper et al (1985) found a low correlation coefficient of .57, but a bias ± precision of 1.4 ± 3.1%. These bias and precision values are similar to those of volunteer studies and the two other adult clinical studies presented in table 1. The clinical studies of Mihm et al (1985) and Cecil et al obtained the values of .8 to .9 which were higher than those of Tremper due to the wider saturation range over which the data were collected. Comparing these results demonstrates how the correlation coefficient alone can be a misleading index of accuracy in methods-comparison studies.

The accuracy of pulse oximeters is impressive, considering the many possible sources of error. We should remember that the specified uncertainty of ± 2% to 3% is for one SD, or a 68% confidence interval. If we desire 95% (2 SD) or 99% (3 SD) confidence, then the uncertainty is two or three times as large, respectively.

Experimental studies :

The pulse oximeter is an excellent monitor for transport from the operating room to recovery because of its portability and ease of use. In a study of American Society of Anesthesiologists Class I and II patients being transported while breathing room air, Tyler and associates found that 35% of their patients exhibited SpO_2 values below 90% during transport. This hypoxemia correlated with obesity and a preoperative history of asthma.

In a related study, Graham and colleagues found that 18 patients transported while breathing room air desaturated to an average SpO_2 of 89%, whereas 19 similar patients transported with supplemental oxygen experienced no major desaturations. In these two studies, the pulse oximeter firmly established the value of supplemental oxygen during transport to the recovery room.

Choi and associates used SpO_2 to monitor post-cesarean section patients who were being treated with either epidural or parenteral narcotics. Each patient was

monitored for approximately 1,000 minutes. Both groups exhibited an average of 3 to 4 minutes of desaturations below 90%, with no significant difference between the groups. The work of Nakatsuka and Rolling (Departments of Anesthesiology and Nursing, Medical College of Virgina, Richmond, Virginia) on the "incidence of post-operative hypoxemia in the recovery room detected by the pulse oximeter" has revealed the important findings of their study. In the 101 post-surgical patients, 12 patients (11.9%) developed moderate hypoxemia ($SaO_2 < 90\%, > 85\%$) and 6 patients (5.9%) suffered severe hypoxemia ($SaO_2 < 85\%$). Smoking habit was significantly associated with post-operative hypoxemia ($P < 0.05$). Supplemental O_2 inhalation decreased the incidence of hypoxemia significantly ($P < 0.03$). There was a trend toward a higher incidence of post-operative hypoxemia in patients with ASA III, chest surgery and ventilator support. Supine head down position and lateral position in the recovery room seemed to have higher incidence of hypoxemia.

New applications for the pulse oximeter are being discovered on a regular basis. A pulse oximeter placed on the great toe has been used as an aid in cannulating the femoral artery in obese patients. The pulse oximeter is now accepted as the primary indicator for and monitor of home oxygen therapy in patients with severe obstructive lung disease.

Clinical Consequences of Pulse Oximetry :

As any new technique becomes standard of care, there is a time window during which it is ethically feasible to perform randomized, controlled studies of its effectiveness. A recent clinical study by Cote' et al (1988) has confirmed the necessity of SpO_2 monitoring during pediatric anesthesia. One hundred and fifty-two patients were continuously monitored with SpO_2 during anesthesia. In half of these patients, the SpO_2 data were "unavailable" to the anesthetic team. A major desaturation event was defined as SpO_2 less than 85% for 30 s or longer. There were 24 major events in 76 cases when SpO_2 data were "unavailable", and only 11 when the SpO_2 data were "available". The majority of these events occurred in patients below 2 yr of age in both groups. Smaller pediatric patients have a greater tendency to desaturate due to their relatively high oxygen consumption, smaller functional residual capacity and possible fetal circulatory pattern. Raemer et al (1987) blindly collected SpO_2 data from 108 out-patients during gynecologic surgery. They found episodes of moderate desaturation ($SpO_2 < 90\%$) in 10% of the cases and severe hypoxemia ($SpO_2 < 85\%$) in 5% of the cases. Under current recommended standards for anesthetic monitoring, it may be difficult to conduct further controlled studies on intra-operative SpO_2 monitoring.

Monitoring during transport to the recovery room has also been examined in children and adults. Pullerits et al monitored 71 healthy pediatric patients during transport and found that 28.1% had SpO_2 values $<$ 90%, while only 45% of these desaturated patients had observable cyanosis. In a similar study of adult patients, Tyler et al found that 35% had SpO_2 values $<$ 90%, and 12% had SpO_2 falls to 85% or less. Both studies conclude that due to the high incidence of desaturation and the inability to clinically recognize it, all patients should receive supplemental oxygen during transport from the operating room to the recovery room.

The oxygenation of adult and pediatric patients in the recovery room has been evaluated with interesting results. Soliman et al compared SpO_2 to a post-anesthesia recovery score in children. The post-anaesthesia recovery (PAR) score is a system based on motor activity, respiratory effort, blood pressure, consciousness, and color. An SpO_2 $>$ 95% was considered adequate oxygenation for a healthy pediatric patient. They found no correlation between the PAR score and the patients' oxygenation. They concluded that pediatric patients in the recovery room should be monitored continuously with pulse oximetry or at least treated with supplemental oxygen regardless of their apparent wakefulness, and that an SpO_2 value should be included among the recovery room discharge criteria.

Morris et al (1986) studied 241 adult patients in the recovery room, measuring SpO_2 values upon arrival, 5 min after arrival, 30 min. after arrival and just prior to discharge. The recovery room personnel were blinded to the SpO_2 data. Of the 149 inpatients studied, 14% had episodes of desaturation to below 90%. As might be expected, the factors associated with desaturation were obesity, extensive surgery, age, and ASA physical status. Most surprising is the fact that more patients were found to be hypoxemic at the time of discharge than at any of the other measurement times. These results demonstrate our present lack of knowledge as to what saturation levels imply immediate danger or a poor prognosis in post-operative patients under various clinical circumstances.

In examining the clinical consequences of pulse oximetry, we must be aware of the pulse oximeter's limitations. Arterial oxygen tension can vary over a wide range during general anesthesia, but SpO_2 will reflect none of this variation until desaturation occurs as Pa_{O_2} decreases below 100 mmHg. The pulse oximeter is effectively a sentry standing at the edge of the "cliff" of desaturation.

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MATERIAL AND METHODS

MATERIAL AND METHODS

The study was conducted in the Gynaecology and Obstetrics Operation Theatre of M.L.B. Medical College Hospital, Jhansi, during the year 1990.

Selection of cases :

The patient requiring abdominal and vaginal hysterectomy operations of A.S.A. Grade 1 & 11, between the age group of 25 to 60 years were selected from the Gynaecology & Obstetrics ward of the M.L.B. Medical College Hospital, Jhansi.

Patient's name, age, body weight and M.R.D. No. were noted and a thorough history and physical examination was done prior to the day of the operation.

Routine investigations like estimation of haemoglobin, blood sugar and blood urea level and routine and microscopic examination of urine were done. Electro-cardiogram and Chest X-ray were done when indicated. The protocol for this study was institutionally approved and written consent was obtained for each patient.

The pulse oximeter used was the Minolta PULSE OX-7. A light source generated by two light emitting diodes (LEDs),

wavelengths at approximately 660 nm and 940 nm and a photodiode (finger probe) was mounted in a finger receptacle. No heating or "arterialisation" technology were required. Circuit control, saturation calculation and display were managed by a micro-processor instrument. No user calibration procedure was required.

Blood pressure recording was done by the Sphigmomanometer instrument.

Each of the cases were examined thoroughly before induction of anaesthesia. Pulse rate, blood pressure, respiratory rate, arterial oxygen saturation by pulse oximeter and mean of three readings of tidal volume and minute volume were recorded.

Premedication :

After establishment of intravenous line with 16 G I.V. Canula, cases were premedicated with Atropine 0.6 mg and Diazepam 5 to 10 mg injected slowly 5 minutes before anaesthesia.

Techniques of Anaesthesia :

In this study, only two anaesthetic techniques were used :

1. General Anaesthesia : $O_2 + N_2O$ + Ether

   $O_2 + N_2O$ + Relaxant.

2. Spinal Anaesthesia.

General Anaesthesia :

In general anaesthesia, preoxygenation was done for atleast 5 minutes, then patients were induced with a sleep dose of 2.5% Sodium Thiopentone (Pentothal) followed by 50 - 100 mg Succinylcholine (Scoline). IPPV started and followed by endotracheal intubation.

For the maintenance of anaesthesia, patients were divided into two groups.

Group A : $O_2 + N_2O$ + Ether

Group B : $O_2 + N_2O$ + Pancuronium Bromide (Pavulon).

Group B patients were on IPPV with the divided doses of Pancuronium and Pentazocin.

Patients were reversed by the Neostigmine 2.5 mg and Atropine 1.2 mg.

Spinal Analgesia :

In spinal analgesia, 1.4 ml of 1% Bupivacaine (Marcaine) by the lumbar puncture needle of 20 G was injected into the subarachnoid space between the L_3 & L_4 space in left lateral or right lateral position under complete aseptic condition. 10^0 head down tilt was given after maintaining the supine position. After the establishment of the block surgery was allowed to proceed in supine position (Abd. Hysterectomy) or Lithotomy position (Vag. Hysterectomy).

Measurement / Assessment :

The pre, per and post-operative evaluation was done by the same person. During operation, pulse, blood pressure, arterial oxygen saturation (SaO_2) by the pulse oximeter, respiratory rate, tidal volume and the subjective assessment of blood loss during operation were recorded.

Post-operative follow-up :

The patients were shifted to recovery room attached to the operation theatre and were watched post-operatively. The pulse rate, blood pressure, arterial oxygen saturation, respiratory rate were recorded in the immediate post-operative period.

Analysis of data :

The results obtained from the three groups of the patients were compared using the simple statistical methods. The Paired 't' test was used to compare the differences between the pre, intra and post-operative values in all the three groups (A, B, C.) and 'P' value was taken from the chart of probability.

Statistical Calculation :

1. Mean $\bar{X} = \frac{X}{n}$

where X = number of frequencies

n = number of patients.

2. Standard Deviation (S.D.) = $\sqrt{\frac{(x - \bar{x})^2}{n}}$

where X = number of frequencies,

$\bar{X}$ = mean

n = number of patients.

3. Degree of freedom (d.f.) = n - 1

4. Standard error of mean = $\frac{S.D.}{\sqrt{n}}$

where SD = standard deviation of mean.

n = number of patients.

5. "Paired t-test"

$$t = \frac{\bar{d}}{sd/\sqrt{n}}$$

where $\bar{d}$ = $\frac{d}{n}$ (d = difference between X & Y)

n = number of patients.

sd = standard deviation of d series.

6. 'p' value = taken from the chart of probability.

OBSERVATIONS

OBSERVATIONS

The present study has been made on a series of 50 cases admitted in M.L.B. Medical College Hospital, Jhansi.

Patients were divided into three different groups according to the anaesthetic technique :

Groups	No.of pts.		Anaesthetic Technique
A	10	:	General Anaesthesia (Ether)
B	10	:	General Anaesthesia (Pavulon)
C	30	:	Spinal Analgesia (Bupivacain).

Table 1

Age distribution of patients in each group.

Age (years)	Study groups					
	A		B		C	
	No.	%	No.	%	No.	%
25 - 34	-	-	2	20.00	6	20.00
35 - 44	6	60.00	4	40.00	10	33.33
45 - 54	2	20.00	4	40.00	10	33.33
55 - 64	2	20.00	-	-	4	13.34
Mean ± S.E.	43.40 ±1.93		40.00 ±1.78		41.93 ±1.87	
Total	10	100.00	10	100.00	30	100.00

Table 1 shows that majority of patients were between 35 - 44 years of age in all the groups. In the age group of 25-34 years in group B, 20% and in group C, 20% patients were present. Between the 35-44 years of age group, in group A - 60%, in group B - 40% and in group C - only 33.33% patients were present. Between the 45-54 years of age group, in group A - 20%, in group B - 40% and in group C - 33.33% patients were present. Between 55-64 years of age group, in group A - 20%, in group B - there was no patient and in group C - 13.34% patients were present.

Table 2

Weight of different groups.

Weight (kg.)	Study groups					
	A		B		C	
	No.	%	No.	%	No.	%
45 - 54	7	70.00	7	70.00	14	46.67
55 - 64	2	20.00	1	10.00	13	43.33
65 - 74	1	10.00	2	20.00	3	10.00
Mean ± S.E.	54.40 ±1.26		53.60 ±1.22		55.90 ±1.35	
Total	10	100.00	10	100.00	30	100.00

The table 2 shows that the average weight of the patients between the 45-54 kg. of weight, in group A - 70%, group B - 70% and in group C - 46.67% patients were present. Between the 55-64 kg. of weight, in group A - 20%, in group B - 10% and in group C - 43.33% patients were present. Between the 65-74 kg. of weight, in group A - 10%, in group B - 20% and in group C - only 10% patients were present. Majority of the patients were between the 45-54 kg. of weight.

Table 3

ASA Grade distribution in different groups.

ASA Grade	Study groups					
	A		B		C	
	No.	%	No.	%	No.	%
I	9	90.00	8	80.00	22	73.33
II	1	10.00	2	20.00	8	26.67
Total	10	100.00	10	100.00	30	100.00

Table 3 shows the grade-wise distribution of the patients which was recommended by the American Society of Anaesthesiologist (ASA).

Majority of the patients were in the grade I. In the ASA grade I, in group A - 90%, group B - 80% and in group C - 73.33% patients were present. In the ASA grade II, in group A - 10%, in group B - 20% and in group C - only 26.67% patients were present.

Table 4

Type of operation in each group.

Types of operation	Study groups					
	A		B		C	
	No.	%	No.	%	No.	%
Abdominal Hysterectomy	5	50.00	5	50.00	19	63.33
Vaginal Hysterectomy	5	50.00	5	50.00	11	36.67
Total	10	100.00	10	100.00	30	100.00

Table 4 shows the type of operation (abdominal or vaginal) in hysterectomy. Majority of the cases were done by the abdominal hysterectomy.

Table 4 shows that the abdominal hysterectomy was performed over the patients in group A - 50%, group B - 50% and in group C - 63.33%. Vaginal hysterectomy was performed over the patients in group A - 50%, in group B - 50%, and in group C - 36.67%.

Table 5

Changes in pulse rate.

Study groups		Operative periods		
		1	11	111
A	Mean ±S.E.	111.5 ±1.59	95.2 ±1.02	94.1 * ±1.25
B	Mean ±S.E.	114.7 ±1.20	93.9 * ±1.40	91.8 * ±1.13
C	Mean ±S.E.	104.7 ±1.26	89.0 ±1.04	87.6 * ±1.06

* $P < 0.05$

In table 5, the operative periods were divided into pre-operative (1), intra-operative (11), and post-operative (111) in different groups.

Table 5 shows that the pulse rate was higher in all the groups in pre-operative period. In the group A - (1) the pulse rate was in the pre-operative period 111.5/mt., in intra-operative period 95.2/mt., and in post-operative period 94.1/mt. In group B, the pulse rate was in the pre-operative period 114.7/mt., in intra-operative period 93.9/mt., and in post-operative period 91.8/mt. In group C, the pulse rate was in the pre-operative period 104.7/mt., in the intra-operative period 89.0/mt. and in the post-operative period 87.4/mt.

In group A the P value was statistically significant in the post-operative period. In group B, the P value was statistically significant in intra and post-operative periods. In group C, the pulse rate was statistically significant during the post-operative period.

Table 6

Changes in respiratory rate in each group.

Study groups		Operative periods I	II	III
A	Mean ±S.E.	19.18 ±0.19	19.20 ±0.19	19.12 ±0.25
B	Mean ±S.E.	16.10 ±0.24	14.20 ±0.26	17.60 ±0.19
C	Mean ±S.E.	18.64 ±0.24	18.60 ±0.24	18.32 ±0.26

$P > 0.05$

Table 6 shows changes in respiratory rate per minute in pre-operative, intra-operative and post-operative periods in different groups. In none of the groups, significant changes could be observed as compared to pre-operative values.

The values were not clinically as well as statistically significant ($P > 0.05$).

<u>Table 7</u>

<u>Changes in Tidal Volume in each group.</u>

Study groups		Operative periods I	II	III
A	Mean ±S.E.	353.2 ±8.13	354.8 ±8.12	363.6* ±8.06
B	Mean ±S.E.	356.8 ±8.05	356.8 ±8.05	353.2 ±8.13
C	Mean ±S.E.	354.8 ±8.12	353.2 ±8.13	354.8 ±8.12

$* P < 0.05$

Table 7 shows significant decrease in the tidal volume in post-operative period (343.6) in group A, as compared with pre-operative tidal volume (353.2). In group B, there is slight decrease in tidal volume (353.2) in post-operative period as compared to pre-operative tidal volume (356.8) but not significant. In group C, there is no change in tidal volume clinically as well as statistically. P value was statistically significant in the post-operative period of the group A.

Table 8

Changes in minute volume (Lit./mt.) in each group.

Study groups		Operative periods		
		I	II	III
A	Mean $\pm$S.E.	6.62 $\pm$0.18	6.63 $\pm$0.18	6.31 $\pm$0.15
B	Mean $\pm$S.E.	6.62 $\pm$0.18	6.31 $\pm$0.15	6.68 $\pm$0.17
C	Mean $\pm$S.E.	6.63 $\pm$0.18	6.62 $\pm$0.18	6.63 $\pm$0.18

P 7 0.05

Table 8 shows minute volume changes in pre-operative, intra-operative and post-operative periods in all the three groups. There were no clinical as well as statistical significant changes observed in all the three groups in all the periods.

<u>Table 9</u>

<u>Changes in mean blood pressure (mm of Hg.) in each group.</u>

Study groups		Operative periods I		II		III	
		Syst.	Diast.	Syst.	Diast.	Syst.	Diast.
A	Mean ±S.E.	117.8 ±1.07	76.00 ±1.03	117.4 ±1.42	76.80 ±0.99	113.0 ±1.20	77.40 ±0.93
B	Mean ±S.E.	120.4 ±1.33	76.60 ±1.00	108.6* ±1.11	76.80 ±0.99	115.6 ±1.16	76.40 ±0.77
C	Mean ±S.E.	124.4 ±1.43	77.46 ±1.20	104.0* ±1.13	76.00 ±0.93	108.0* ±1.22	76.40 ±0.97

* $P < 0.05$

Table 9 is showing the mean blood pressure (mm of Hg.) changes in pre-operative, intra-operative and post-operative periods in all the three groups. In group A there were no significant changes in mean blood pressure, in intra-operative and post-operative periods as compared with the pre-operative mean blood pressure. In group B, there were significant fall in mean blood pressure in intra-operative

period (108.4/76.80) as compared with the pre-operative mean blood pressure (120.4/76.60). But there was no significant change in the mean blood pressure in post-operative period (115.6/76.40). In group C, there were significant fall in mean blood pressure in intra-operative period (104/76) and post-operative period (108/76.40) as compared with the pre-operative mean blood pressure (124.4/77.46). The values were clinically as well as statistically significant ($P < 0.05$) in group C.

Table 18

Haemoglobin (Hb%) of patient in each group.

Hb (gm%)	Study groups					
	A		B		C	
	No.	%	No.	%	No.	%
8 - 9	1	10.00	-	-	7	23.33
10 - 11	9	90.00	7	70.00	10	33.34
12 - 13	-	-	1	10.00	7	23.33
14 - 15	-	-	2	20.00	6	20.00
Total	10	100.00	10	100.00	30	100.00

Table 10 is showing the Haemoglobin (gm%) level of patients in each group. In group A, there were 10% patients in the range of 8-9 gm% and 90% patients were in the range of 10-11 gm%. None of the patient was in the range of 12-13 or 14-15 gm%.

In group B, there were none of the patients in the range of 8-9 gm%. There were the 70% patients in the range of 10-11 gm% and 10% patients in the range of 12-13 gm% and 20% patients were in the range of 14-15 gm%.

In group C, there were 23.33% patients in the range of 8-9 gm% and 33.34% patients in the range of 10-11 gm% and 23.33% patients were in the range of 12-13 gm% and 20% patients were in the range of 14-15 gm%.

Table 11

Arterial oxygen saturation (SaO_2%) in different groups.

Study groups		SaO_2 (%) I	II	III
A	Mean ±S.E.	97.60 ±1.42	96.60 ±1.28	91.80* ±1.20
B	Mean ±S.E.	97.20 ±1.33	98.30 ±1.14	95.10 ±1.11
C	Mean ±S.E.	97.36 ±1.03	87.46* ±1.07	89.36* ±0.99

* $P < 0.05$

Table 11 is showing the arterial oxygen saturation (SaO_2 %) in pre-operative, intra-operative and post-operative periods in all the three groups. In group A, there was significant fall in arterial oxygen saturation in post-operative period (91.80) as compared with the pre-operative (97.60) arterial oxygen saturation. In group B, there was no significant change in the arterial oxygen saturation in the arterial oxygen saturation in the intra-operative period as well as in the post-operative period. In group C, there were significant fall in the arterial oxygen saturation in intra-operative period (87.46) and post-operative period (89.36) as compared with the pre-operative (97.36) arterial oxygen saturation. The values were clinically as well as statistically significant ($P < 0.05$) in the group C.

Table 12 is showing the effect of the pulse rate, blood pressure and respiratory rate over the arterial oxygen saturation in intra-operative as well as the post-operative periods.

In intra-operative period of group A, there were no significant effect of pulse rate, mean blood pressure and respiratory rate over the arterial oxygen saturation. In group B, there was the significant decrease in the pulse rate (93.9), mean blood pressure (from 120.4/76.00 to 108.6/76.80), but no significant effect over the arterial oxygen saturation. Pulse rate and respiratory rate were also not effecting the arterial oxygen saturation.

Table 12

Effect of Pulse rate, B.P. and respiratory rate over the SaO_2 % in different groups of the patients during the intra and post-operative periods. All the values are in the mean value.

Study groups	Pulse rate/mt.	B.P. (mm Hg.)	Resp. rate/mt.	SaO_2 (%)	
A	111.5	117.8/76.00	19.18	97.60	I Pre-operative
B	114.7	120.4/76.60	16.10	97.20	
C	104.7	124.4/77.46	18.64	97.36	
A	95.2	117.4/76.80	19.28	96.80	II intra-operative
B	93.9*	108.6/76.80*	14.20	98.30	
C	89.0	104.0/76.00*	18.60	87.46*	
A	94.1*	113.0/77.40	19.12	91.80*	III post-operative
B	91.8*	115.6/76.40	17.80	95.10	
C	87.6*	108.0/76.40*	18.32	89.36*	

* $p < 0.05$

In group C, when arterial oxygen saturation was significantly decreased (87.46) as compared with the pre-operative period (97.36), there were no significant effect in the pulse rate but decrease in the mean blood pressure (from 124.4/77.46 to 104.0/76.00). There was slight decrease in the respiratory rate but not significantly.

In post-operative period of group A, there was significant fall in the arterial oxygen saturation (91.80) as compared with the pre-operative (97.60) arterial oxygen saturation. There were significant decrease in the pulse rate (from 111.5 to 94.1) but no significant fall in mean blood pressure (from 117.8/76.00 to 113.0/77.40) and respiratory rate (from 19.18 to 19.12). In group B, there were the significant fall in the pulse rate (from 114.7 to 91.8) but no significant effect of the mean blood pressure and respiratory rate over the arterial oxygen saturation. In group C, when arterial oxygen saturation was significantly decreased (89.36) as compared with the pre-operative (97.36) arterial oxygen saturation there were also significant decrease in the pulse rate (from 104.7 to 87.6), mean blood pressure (from 124.4/77.46 to 108.0/76.40) and slight decrease in the respiratory rate (from 18.64 to 18.32) but not statistically significant.

Table 11

Effect of Hb% over the SaO_2% during the intra and post-operative periods in different groups of the patients. All values are mean values.

Study groups	Hb (gm%)	SaO_2 % I	SaO_2 % II	SaO_2 % III
A	8 - 9	96.00	94.00	86.00*
	10 - 11	97.80	96.89	92.44
	12 - 13	-	-	-
	14 - 15	-	-	-
B	8 - 9	-	-	-
	10 - 11	97.00	98.43	94.43
	12 - 13	96.00	96.00	95.00
	14 - 15	98.50	99.00	98.00
C	8 - 9	97.29	82.14*	85.43*
	10 - 11	97.50	87.90*	89.70
	12 - 13	97.57	91.57	92.86
	14 - 15	97.33	88.17*	89.33

* $P < 0.05$

Table 13 is showing the effect of Hb% over the arterial oxygen saturation in intra-operative and post-operative periods in all the three groups.

In post-operative period of the group A, when the arterial oxygen saturation was significantly decreased (86.00) as compared to pre-operative period (96.00), the Hb% of the patients were 8 - 9 gm%. In the range of 10-11 gm% of Hb there were no significant change in the arterial oxygen saturation.

In group B, there were no significant effect of the Hb% level over the arterial oxygen saturation during the intra and post-operative periods.

In group C, there were the significant decrease in the arterial oxygen saturation in intra-operative (82.14) and post-operative (85.43) period as compared with the pre-operative (97.29) arterial oxygen saturation in the range of 8-9 gm% of Hb. There was the significant decrease in the intra-operative arterial oxygen saturation (87.90) as compared with the pre-operative (97.50) saturation in the range of 10-11 gm% of Hb. There were no significant effect of the Hb in the range of 12-13 gm% over the arterial oxygen saturation during the intra and post-operative periods. In the range of 14-15 gm% Hb, there were the significant fall in the arterial oxygen saturation during the intra-operative period but no significant effect in the post-operative period.

Table 14

Effect of type of operation over the arterial oxygen saturation (SaO_2%) (Abdominal or Vaginal).

Study groups	Type of operation		SaO_2% (Mean value) I	II	III
A	Abdominal Hysterectomy	Mean ±S.E.	97.80 ±1.11	96.40 ±1.07	91.60* ±1.03
	Vaginal Hysterectomy	Mean ±S.E.	97.60 ±1.07	96.80 ±0.99	92.40 ±0.97
B	Abdominal Hysterectomy	Mean ±S.E.	97.20 ±1.03	98.40 ±0.93	95.20 ±1.00
	Vaginal Hysterectomy	Mean ±S.E.	97.20 ±1.22	98.20 ±1.21	95.00 ±1.28
C	Abdominal Hysterectomy	Mean ±S.E.	97.42 ±1.33	88.58* ±1.28	90.26* ±1.42
	Vaginal Hysterectomy	Mean ±S.E.	97.27 ±1.03	85.58* ±0.97	87.73* ±0.99

* $P < 0.05$

Table 14 is showing the effect of the type of operation over the arterial oxygen saturation in intra-operative and post-operative period of all the three groups.

In group A, there was the significant fall in the arterial oxygen saturation in the post-operative period (91.60) as compared to the pre-operative arterial oxygen saturation (97.80) when abdominal hysterectomy operation was performed. In vaginal hysterectomy operation, there were no significant changes.

In group B, no significant changes were observed in the intra-operative period and post-operative period in both types of operations - abdominal as well as vaginal hysterectomy.

In group C, when abdominal hysterectomy operation was performed, there were significant fall in the arterial oxygen saturation in intra-operative period (88.58) and post-operative period (90.26) as compared with the pre-operative (97.42) arterial oxygen saturation. When vaginal hysterectomy operation was performed, there were also significant fall in the arterial oxygen saturation in intra-operative (85.58) and post-operative (87.73) periods as compared with the pre-operative (97.27) arterial oxygen saturation. The values were clinically as well as statistically significant ($P < 0.05$) in group C in both types of operation i.e. abdominal or vaginal hysterectomy.

<u>Table 12</u>

<u>Arterial oxygen saturation (SaO_2%) during the immediate post-operative period in different groups</u>.

Study groups	SaO_2 %		
	$> 90\%$	$< 90\% - > 85\%$	$< 85\%$
A n = 10	7	3	-
B n = 10	10	-	-
C n = 30	15	11	4

Table 15 shows the arterial oxygen saturation of the patients during the immediate post-operative period in all the three groups.

In group A, out of the 10 patients, there were 7 patients having the arterial oxygen saturation more than 90% and 3 patients were having the arterial oxygen saturation between 90% and 85%, none of the patient was having less than 85%.

In group B, all the patients were having the arterial oxygen saturation more than 90%.

In group C, out of the 30 patients, there were 15 patients having the arterial oxygen saturation more than 90%, 11 patients were having between 90% and 85% and 4 patients were having their arterial oxygen saturation less than 85%.

In group C, only the patients were supplemented by intra-nasal O_2 inhalation in post-operative period.

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D I S C U S S I O N

DISCUSSION

Hypoxemia has been a matter of concern not only in the operating room but also in the immediate post-operative period. However, clinical assessment of hypoxemia is rather difficult. Cyanosis is only detectable when the arterial oxygen saturation ($Sa\ O_2$) is below 80% (M. Nakatsuka et al, 1989). The human eye is a poor judge of changes in skin colour, particularly in dark-skinned patients and under fluorescent lights. The recent introduction of pulse oximetry has provided a continuous, non-invasive, real time method to detect SaO_2 intra-operatively and post-operatively (M. Nakatsuka et al, 1989). Anaemia is very frequently seen in Indian women and anaesthetic practice moreso with major surgery like hysterectomy operations.

The present study was conducted in the series of 50 patients undergoing hysterectomy with three different anaesthetic techniques. Continuous SaO_2 monitoring was done with the pulse oximeter (Minolta PULS-OX-7) and following clues were made.

In our observations the SaO_2 was not affected by the age and weight of the patient as also confirmed by M. Nakatsuka and D. Rolling (1989) who had shown that the

age and weight had no significant effect on the post-operative hypoxemia.

The SaO_2 was significantly affected by the type of operation (Table 14). Under general anaesthesia with Ether, there was significant decrease in the SaO_2 during the post-operative period (91.60%) as compared with the pre-operative period (97.80%) in abdominal hysterectomy operation. Under general anaesthesia with muscle relaxant, there was no significant effect over the SaO_2 during the intra and post-operative periods. There was significant fall in SaO_2 in patients undergoing spinal analgesia which responded very well to the oxygen therapy. The finding is not in accordance with the study of M. Nakatsuka and D. Bolling (1989) who didn't find any significant changes in different anaesthesia techniques. Patients undergoing vaginal hysterectomy had more desaturation (pre-operative 97.27%, intra-operative 85.55% and post-operative 87.73%) as compared to patients undergoing abdominal hysterectomy (pre-operative 97.42%, intra-operative 85.58% and post-operative 90.26%). This might be due to the lithotomy and head down position in vaginal surgery as confirmed by the study of M. Nakatsuka and D. Bolling (1989).

Morris et al studied 241 adult patients in the recovery room, measuring SpO_2 values upon arrival, 5 min. after arrival, 30 min. after arrival and just prior to discharge. The recovery room personnel were blinded

to the SpO_2 data. Of the 149 inpatients studied, 14% had episodes of desaturation to below 90%. As might be expected, the factors associated with desaturation were obesity, extensive surgery, age and ASA physical status. Most surprising is the fact that more patients were found to be hypoxemic at the time of discharge than at any of the other measurement times. These results demonstrate our present lack of knowledge as to what saturation levels imply immediate danger or a poor prognosis in post-operative patients under various clinical circumstances.

The SaO_2 was significantly affected by the fall in blood pressure (20 - 30%) during the intra and post-operative periods. The episodes of desaturation was seen in the patients undergoing the hysterectomy operation under the spinal analgesia. After 10 to 15 mts. of giving the spinal analgesia there was the fall in blood pressure due to the sympathetic block of the lower half of the body. Due to the reduction in the blood pressure there was the tissue hypoxia or lowered arterial oxygen concentration towards the peripheral tissue. This episode was detected by the finger probe and oximeter shown the fall in the SaO_2. The SaO_2 was not affected significantly in those patients who were going to be operated under general anaesthesia during the intra-operatively as well as the post-operatively. This was only because of the adequate administration of the gas mixture and ventilation during the intra-operative period. In post-operative period these patients were not

significantly affected as the oximeter shown little difference in the pre-operative and post-operative readings of the SaO_2 (Table 9 and 12).

The SaO_2 was significantly affected by the pulse rate during the intra and post-operative periods (Table 5 and 12). The SaO_2 was decreased when pulse rate was decreased. It was seen that when blood pressure was decreased there was increase in the pulse rate for few seconds and later on pulse rate was decreased. Due to the fall in the blood pressure and pulse rate there was the reduced cardiac output. Peripheral tissue perfusion was affected significantly according to the post-operative readings of the oximeter. On the contrary, the study carried out by M. Nakatsuka and D. Bolling (1989) shown that "the anaesthesia technique had no significant effect on post-operative hypoxemia. There was a trend towards a higher incidence of post-operative hypoxemia in supine head down position of the patients in the recovery room".

The SaO_2 was significantly affected by the respiratory rate during the intra-operative and post-operative periods (Table 6 and 12). In the study, the SaO_2 was significantly decreased when the respiratory rate was decreased. In those patients who were going to be operated under spinal analgesia were not affected significantly during the post-operative period. After giving sedation (Diazepam 5 - 10 mg) to the patient,

there was a fall in the respiratory rate, there was a fall in the SaO_2 during the intra-operative as well as the post-operative periods. The patients undergoing hysterectomy with general anaesthesia with ether were affected more rather than the patients undergoing hysterectomy under general anaesthesia with muscle relaxant. This was only because of the residual effect of the ether. Peripheral vasoconstriction could be a contributing factor over the patients during the post-operative period. Patients were not able to breath satisfactorily in the recovery room due to the post-operative pain. But the patients undergoing the hysterectomy under general anaesthesia with the muscle relaxant were reversed adequately and patients were fully conscious. The conscious patients were able to breath satisfactorily during the post-operative periods without any pain because of the effect of analgesics. As it was confirmed by the study of M. Nakatsuka and D. Bolling (1989) that the use of muscle relaxant had no significant effect on hypoxemia.

There was the remarkable effect of the hemoglobin level over the SaO_2 (Table 10 and 13). It was seen that the SaO_2 was decreased significantly in the low hemoglobin level patients during the intra as well as the post-operative periods. In this study, the patients who were having hemoglobin level less than 10 gm% were having the

SaO_2 below 90% during the post-operative period. This was only because of the poor compensatory mechanisms for the blood lost during operation. Lower level of the hemoglobin directly affect the oxygen demand of the tissues and patients developed tissue hypoxia. Due to the tissue hypoxia, the SaO_2 was also reduced and patients developed hypoxemia post-operatively. The study of M. Nakatsuka and D. Bolling (1989) suggest that there was a trend towards a higher incidence of post-operative hypoxemia in patients with ASA III, chest surgery and ventilator support.

This study demonstrated that the SaO_2 was significantly affected by the fall in the blood pressure, pulse rate, respiratory rate and low hemoglobin level of the patients during the intra-operative as well as the post-operative periods. It was seen that the SaO_2 was significantly affected in those patients who were operated under the spinal analgesia with bupivacaine. The patients who were operated under the general anaesthesia with Ether were also having the low SaO_2 but not significantly. The patients undergoing hysterectomy operation under general anaesthesia with the muscle relaxant were having the adequate arterial oxygen saturation during the intra-operative as well as the post-operative periods. The study of Nakatsuka and Bolling (1989) confirmed that supplemental O_2 inhalation

decreased the incidence of hypoxemia significantly. "There was a trend towards a higher incidence of post-operative hypoxemia in patients with ASA III, chest surgery and ventilator support. Supine head-down position and lateral position in the recovery room seemed to have higher incidence of hypoxemia. Age, anaesthesia technique, use of muscle relaxant, body temperature, post-anaesthesia medication, obesity, history of asthma, COPD, RLD and heart disease had no significant effect on hypoxemia".

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CONCLUSION

CONCLUSION

In this study, the conclusion was derived that -

1. The anaemic patients of less than 10 gm Hb% were more prone to developed post-operative hypoxemia particularly during the post-operative period more common when done under spinal analgesia.

2. The bradycardia reduces the SaO_2% during the intra- as well as the post-operative periods.

3. The hypotension reduced the SaO_2% during the intra as well as the post-operative periods.

4. The fall in respiratory rate reduced the SaO_2%.

5. The desaturation was more in the spinal analgesia rather than the general anaesthesia.

6. The fall in the SaO_2% was more in the patients undergoing hysterectomy under general anaesthesia with Ether rather than the patients undergoing hysterectomy under general anaesthesia with muscle relaxant.

7. The SaO_2% was greatly reduced in the Lithotomy, Trendelenburg position.

8. The supplemental O_2 inhalation during the intra and post-operative periods greatly reduced the incidence of post-operative hypoxemia.

In conclusion, we recommend that the hysterectomy operation should be performed under general anaesthesia with muscle relaxant (Pavulon) preferred over inhalational agent (Ether) and spinal analgesia and patients should have hemoglobin level more than 10 gm% and supplemental O_2 inhalation should be done post-operatively in supine position.

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S U M M A R Y

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PULSE OXIMETRY has been recommended as a standard of care for every general anaesthetic. This technique, virtually unknown in anaesthesia 5 years ago, has been so readily adopted for several reasons. The device provides valuable data regarding blood oxygenation and this information is obtained easily, continuously and non-invasively. Continuous assessment of arterial oxygenation is important in clinical management of critically ill or anaesthetized patients.

Hypoxemia has been a matter of concern not only in the operating room but also in the immediate post-operative period. However, clinical assessment of hypoxemia is rather difficult and cyanosis is only detectable when the arterial oxygen saturation (SaO_2) is below 80%. The recent introduction of "PULSE OXIMETRY" has provided a continuous, non-invasive, real time method to detect SaO_2 intra-operatively and post-operatively. Indian women are frequently suffer from anaemia. The risk factors of intra-operative and post-operative hypoxemia in "Hysterectomy" operations with pre-existing anaemia under different techniques of anaesthesia may be injurious to the patients.

Pulse oximetry functions by positioning any pulsating arterial vascular bed between a two wavelength (660 nm & 940 nm Red & infra-red) light source and a detector. The pulsating vascular bed by expanding and relaxing, creates a change in the light path length that modifies the amount of light detected. The familiar plethysmograph wave-form results. Because the detected pulsatile wave-form is produced solely from arterial blood, using the amplitude at each wavelength and Beer's Law allows exact beat-to-beat continuous calculation of arterial hemoglobin saturation with no interference from surrounding venous blood, skin, connective tissue or bone.

"On many occasions this instrument has detected anoxemia when observations of pulse, blood pressure and colour of the patient and peripheral vascular tone have shown no abnormalities".

The clinical utility of the non-invasive oximeter in the operating room was discovered in 1980s by William New, an anaesthesiologist at Stanford University, realising that a continuous, non-invasive monitor of oxygenation would be useful to anaesthesiologists.

As with any new technique, many people contributed to the development of usable in vivo oximeters. Matthes is often considered the father of Oximetry. Between 1935 and

1944 he published a series of articles investigating oxygen transport to tissue by light transmission techniques.

The detection of cyanosis, the traditional sign of hypoxaemia, is very unreliable. The human eye is a poor judge of changes in skin colour, particularly in dark skinned patients and under fluorescent light. Severe arterial hypoxaemia may occur even during the most meticulously administered anaesthetic. Prolonged moderately severe hypoxaemia may be associated with pre-existing anaemia and respiratory disease. Anaemia is very frequently associated with Indian women and anaesthetic practice more so when major surgery like hysterectomy operations. It was therefore thought worthwhile to evaluate changes in oxygen saturation by Pulse Oximetry intra-operatively and in immediate post-operative period in hysterectomy operations planned under different anaesthetic techniques.

The present study was conducted in the series of 30 patients undergoing hysterectomy with three different anaesthetic techniques. Continuous SaO_2 monitoring was done with pulse oximeter (Minolta PULSE-OX-7).

In this study the conclusion was derived that -

- The anaemic patients of less than 10 gm% Hb were more prone to developed post-operative hypoxemia particularly during the post-operative period more common when done under spinal analgesia.

- The desaturation was more in the patients with bradycardia, hypotension and fall in respiratory rate.
- The desaturation was more in the spinal analgesia rather than the general anaesthesia.
- The SaO_2 % was greatly reduced in the Lithotomy, Trendelenburg's position.
- The supplemental O_2 inhalation during the intra and post-operative periods greatly reduced the incidence of post-operative hypoxemia.

In conclusion, we recommend that the hysterectomy operation should be performed under general anaesthesia with muscle relaxant (Pavulon) preferred over inhalational agent (Ether) and spinal analgesia and patients should have hemoglobin level more than 10 gm% and supplemental O_2 inhalation should be done post-operatively in supine position.

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